

# Posttraumatic Stress Disorder: Anxiety or Traumatic Stress Disorder?

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*The authors examine the question of whether posttraumatic stress disorder (PTSD) should continue to be classified with the anxiety disorders in the upcoming revision of the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-V; American Psychiatric Association) classification system. They examine four areas of research that challenge the placement of PTSD among the anxiety disorders: research on peritraumatic emotions and their association with later PTSD symptoms, the role of emotions over the course of PTSD, physiological reactivity and emotional responses, and comorbidity patterns. The authors conclude with the recommendation that PTSD be included among a new category of traumatic stress disorders in DSM-V.*

Principles of diagnostic taxonomy suggest that disorders within a class should share more common features and covary with each other to a greater extent than with disorders from another class. However, recent research on the structure and phenomenology of the anxiety disorders, which are defined by their pathological fear, anxiety, and avoidance symptoms, has raised fundamental questions about their organization and composition and may have implications for the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (DSM-V; American Psychiatric Association [APA]) revision process (cf. Watson, O'Hara, & Stuart, 2008). For example, studies suggest that generalized anxiety disorder may have more in common with major depression and dysthymia than with the other anxiety disorders (e.g., Krueger, 1999; Slade & Watson, 2006; Vollebergh et al., 2001). Other research has shown that obsessive-compulsive disorder shares essential features with a variety of nonanxiety disorder syndromes and may be better located in its own class of "obsessive-compulsive spectrum disorders" (Hollander & Benzaquen, 1997). Similar questions have been raised about posttraumatic stress disorder (PTSD), which has been conceptualized and classified as an anxiety disorder since its inception in *DSM-III* (APA, 1980), but is distinguished from other disorders by virtue of the fact that its definition specifies a causal relationship between exposure to an adverse life event and subsequent symptom development. Concerns about the placement of PTSD among the anxiety disorders are longstanding, as noted in the introduction to the anxiety disorders chapter of *DSM-III-R* (APA, 1987) which read, "The classification of Post-traumatic Stress Disorder is controversial since the predominant symptom is

the reexperiencing of a trauma, not anxiety or avoidance behavior" (p. 235).

The aim of this article is to revisit the question of whether PTSD is best classified as an anxiety disorder and, if not, where it might be better located in *DSM-V*. Our review of the literature supports the following conclusions: (a) fear is just one of many emotions experienced by trauma survivors and is not necessary for the development of PTSD, (b) emotions other than fear or anxiety play a prominent role in the maintenance of PTSD, (c) laboratory studies suggest that reactivity to trauma-related cues often does not reflect pathological fear or anxiety, and (d) findings of comorbidity studies are inconsistent with the current placement of PTSD among the anxiety disorders. We conclude by suggesting that PTSD belongs in its own class of traumatic stress disorders precipitated by exposure to serious adverse life events.

## PROBLEMS WITH PLACING PTSD AMONG THE ANXIETY DISORDERS

### Fear is Just One of Many Common Peritraumatic Emotions

Criterion A of the *DSM-IV PTSD* (APA, 1994) diagnosis specifies that a person must have "experienced, witnessed, or been confronted with an event that involves actual or threatened death or injury, or a threat to the physical integrity of self or others" (p. 467, Criterion A1) and the person must have a subjective emotional response that involved "intense fear, helplessness, or horror" (p. 467, Criterion A2). From *DSM-III* to *DSM-IV*, revisions were made to

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include in the first PTSD criterion not just the seriousness of the traumatic stressor (rather than its rarity), but also one's subjective response to the stressor. However, only three subjective reactions have been listed: fear, helplessness, or horror. The latter two were added to recognize that some people who experience very traumatic events may not necessarily experience fear. For example, a child enduring sexual abuse may experience helplessness, but not fear, *per se*. First responders to a catastrophe (or graves registration workers, medics, etc.) may feel horrified by sensory experiences, but not personally threatened or fearful. By limiting Criterion A2 to fear, helplessness, and horror, the *DSM-IV* does not consider other emotions such as anger and rage, which are experienced during fight rather than flight responses (e.g., as may occur in combatants), shame if an individual is made to endure degradation, or guilt if the trauma involves a violation of one's personal code of conduct or morals.

Criteria A1 and A2 have been the focus of considerable research and criticism (cf. Weathers & Keane, 2007), but most studies that have addressed the A2 criterion have examined its predictive validity, i.e., by evaluating whether A2 provides predictive information about who goes on to develop PTSD beyond that provided by A1, and how the presence or absence of A2 affects PTSD prevalence rates (e.g., Adler, Wright, Bliese, Eckford, & Hoge, 2008; Bedard-Gilligan & Zoellner, 2008; e.g., Breslau & Kessler, 2001). Very few studies have focused on the content validity of the A2 criterion or addressed the question of whether fear, helplessness, and horror provide adequate coverage of the range of peritraumatic emotions that are linked to the subsequent development of PTSD. Studies that have addressed this issue raise questions about the centrality of peritraumatic fear in the development of PTSD. For example, Roemer, Orsillo, Borkovec, and Litz (1998) compared retrospective reports of peritraumatic fear, helplessness, horror, and numbing in trauma-exposed college students and the relationship between these ratings and PTSD symptoms. Results showed that peritraumatic fear and horror were not significantly related to PTSD severity whereas ratings of helplessness and numbing were.

In one of the few prospective examinations of the predictive validity of A2, Brewin, Andrews, and Rose (2000) examined reports of fear, helplessness, horror, and anger as well as other emotions in crime victims within a month after the crime and evaluated whether the presence of fear, helplessness, or horror were uniquely associated with PTSD 6 months later. All three Criterion A2 reactions were associated with PTSD at 6-months posttrauma, but anger at others and shame were also significant independent predictors of PTSD at 6 months. Those few people who developed PTSD but did not report peritraumatic fear, helplessness, or horror all reported either extreme shame or anger at others during the trauma.

Unlike many of the studies on peritraumatic emotions that have been conducted, Resick (2008) did not assess peritraumatic emotions years retrospectively, but 2 weeks after a rape or physical assault, which should reduce concerns about retrospective recall bias. The women were interviewed about their recent event and

were asked to rate their emotions during the incident using a list of emotions adapted from early trauma research (Veronen, Kilpatrick, & Resick, 1979) and included in the *DSM-IV* field trial (Kilpatrick et al., 1998). The women were asked to rate how much of the time they experienced these 18 emotions from 0 (*none of the time*) to 4 (*all of the time*). The 198 women reported a large range of emotions during their traumatic events. When regressed against PTSD severity at 2 weeks postcrime, the set of emotions accounted for 35% of the variance in PTSD symptoms; however, peritraumatic guilt was the only significant independent predictor, (although there were trends for terrified, helpless, angry, detached, and numb). A second analysis was conducted to examine whether the peritraumatic emotions that were assessed at 2-weeks post-trauma predicted PTSD severity at 3 months after accounting for 2-week PTSD. There was a trend ( $p < .07$ ) for the peritraumatic emotions to predict PTSD severity 3 months later, with only a trend for angry to predict 3-month PTSD. Indicators of anxiety (terrified, anxious, afraid) were not significant predictors of PTSD severity at either point of time.

Rizvi, Kaysen, Gutner, Griffin, and Resick (2008) combined this data set with another similar study to examine peritraumatic responses among nearly 300 female rape and assault victims who were assessed for the first time an average of 20 days after the traumatic event. The emotional and behavioral responses were factor analyzed. The first factor, accounting for 24% of total variance, consisted of negative peritraumatic emotions: hurt, sad, betrayed, humiliated, angry, embarrassed, disgusted, guilty, and shocked/surprised. The second factor, a fear factor accounting for 11% of the total variance consisted of afraid, terrified, worried, helpless, and anxious. The last two factors (6% and 5% of total variance respectively) represented active responses and freezing. All of the factors, except active responding were correlated with PTSD and depression. In a regression analysis to predict PTSD symptom severity, the four factors significantly improved the model over time since trauma, type of trauma, and dissociation. Rape, more dissociation, fewer active responses, and greater freeze responses were uniquely associated with higher PTSD symptom severity. Again, fear was not a significant independent predictor of PTSD symptomatology.

In sum, although some studies that have examined peritraumatic fear/anxiety responses have found them to be predictors of PTSD severity, when other peritraumatic emotions or responses are also examined, fear does not appear to play the sole or primary role in the development of the disorder. The next question is whether fear and anxiety play the predominant role in the development and maintenance of chronic PTSD.

## Emotions and the Maintenance of Posttraumatic Stress Disorder

Despite the fact that guilt and shame are not included among the core symptoms of PTSD and anger is only an indicator of

hyperarousal in the *DSM-IV* PTSD symptom criteria, a growing number of studies have implicated these specific emotions as central to the psychopathology of PTSD. Studies have found guilt to be strongly associated with PTSD among combat veterans, battered women, and rape victims (Beckham, Feldman, & Kirby, 1998; Henning & Frueh, 1997; Kubany et al., 1995, 1996; Nishith, Nixon, & Resick, 2005). Leskela, Keiperling, and Thuras (2002) found former prisoners of war with PTSD were more likely to have shame, but not guilt. Street and Arias (2001) also found that in victims of intimate partner violence, shame but not guilt was a powerful predictor of PTSD. Wong and Cook (1992) compared three clinical veterans groups on shame: PTSD, substance abusers, and those with depression. On the shame subscales, both the depressed and PTSD group differed from the substance abusing group on a measure of inferiority whereas PTSD differed from the other groups on a measure of alienation. Andrews, Brewin, Rose, and Kirk (2000) studied shame, anger at self, and anger at others at 1 and 6 months posttrauma along with a range of control variables among victims of violent crime. They found that at 1 month, only shame and anger at others were associated with PTSD severity. They also examined whether these emotions predicted 6-month PTSD after controlling for 1-month PTSD levels. Only shame was an independent predictor of later PTSD severity.

Research focusing specifically on anger has been conducted across different populations. Orth and Weiland (2006) conducted a meta-analysis of 39 studies and found anger to be associated with PTSD, especially in military populations. Riggs, Dancu, Gershuny, Greenberg, and Foa (1992) found that anger among female rape or assault victims was positively related to the development of PTSD. In a prospective study of PTSD, they found that 1 week and 1 month after an assault, female crime victims reported more anger than nonvictimized women and that anger elevation at 1 week was predictive of PTSD severity 1 month later. Those who dropped out of the longitudinal study reported greater inward-focused anger than those who remained in the study. When they divided the victimized group at 1 month by PTSD status, they found that those who had PTSD at 1 month had higher inward-focused anger at 1 week. The non-PTSD victims were no different on anger than the nonvictims.

In a second prospective report from the same study, Feeny, Zoellner, and Foa (2000) examined the participants who met PTSD symptom criteria immediately. By 4-weeks postcrime, the time at which PTSD can be diagnosed, anger predicted PTSD at 12 weeks after controlling for PTSD severity. Ehlers, Mayou, and Bryant (1998), also found that initial anger reactions predicted PTSD severity at 3 months and 1 year later among a large sample ( $N = 967$ ) of motor vehicle accident victims. Because the correlations increased over time, they suggested that anger may play more of a role in maintaining PTSD than its initial development.

Some studies have included more than one emotional response and have examined other ways of assessing emotions than through the use of self-report measures. For example, Reynolds and Brewin

(1999) examined the intrusive experiences of a matched sample of traumatized individuals who were either depressed only or had PTSD (with or without depression). The majority of both groups had experienced intrusive memories in the past week that were then detailed and compared. The first or second most frequent emotions associated with those memories were, in order: anger, sadness, fear, helplessness, and guilt. The PTSD group mentioned helplessness significantly more often than the depressed group. There were no other differences between the groups.

Holmes, Grey, and Young (2005) examined intrusive experiences that were elicited as part of an initial assessment session and then examined the "hot spots" that emerged later during exposure therapy to see if they were similar. After the initial exposure session, the patients ( $n = 32$ ) were asked to identify the worst moments during their recall of the traumatic event (the sample had experienced a range of traumatic events). The patients were also asked to identify the emotions and thoughts that were associated with these memories. They found that over 75% of the time, the hotspots during therapy matched the previous list of intrusive recollections. Emotions words were coded and classified by type. Of 112 emotion words that were used, fear, helplessness, or horror comprised 42%. The remainders were of other emotions such as sadness, anger, or shame. So, even in the context of exposure therapy, the most emotion-laden memories, which are linked to their PTSD reexperiencing symptoms, are associated with fear, helplessness, or horror less than half of the time.

## Reactions to Trauma-Related Cues may not Reflect Fear or Anxiety

Laboratory protocols often ask participants to rate a range of emotions during assessments in which they are presented with trauma-related photos, sounds, or script-driven imagery. In many cases, the ratings of various emotions have been collapsed into negative, positive, or neutral emotional valence, but in some studies, the various emotion ratings were analyzed separately, which allows for an examination of fear compared to other emotions. For example, in one of the earliest PTSD psychophysiological studies, Pitman, Orr, Forgue, de Jong, and Claiborn (1987) asked veterans with current PTSD or who never had PTSD to rate their emotional responses to individualized trauma scripts. Veterans with PTSD reported greater anger, sadness, disgust, and fear, in that order. In a similar study comparing combat veterans with PTSD to those who had never had PTSD but had another anxiety disorder, Pitman et al. (1990) asked veterans to rate their emotional reactions to combat scripts. The PTSD group reported not only greater fear than the anxiety control group, but more sadness, surprise, anger, and disgust.

Carson et al. (2000) assessed Vietnam nurses with or without PTSD from observing/witnessing events with individualized and standardized scripts. Like the previous study with older veterans,

there were no differences between the PTSD and non-PTSD nurses with regard to the emotions they experienced during the physiological assessment of their worst trauma. However, as with the other studies, they reported a range of emotions—with sadness rated highest, followed by anger and disgust. McDonagh-Coyle et al. (2001) conducted psychophysiological assessments with 37 adult survivors of child sexual abuse. They compared responses across pleasant or trauma scripts, and mental arithmetic. There were physiological and emotional differences between the different prompt conditions. The 10 emotion ratings were reduced to five through factor analysis: negative emotions (anger, anxious, ashamed, disgusted, fearful, and powerless), and single items (excited, happy, numb, and sad). Following the trauma script, negative emotions were positively correlated with those who had higher Clinician-Administered PTSD Scale (CAPS) scores (Blake et al., 1995; Weathers, Keane, & Davidson, 2001).

In each of these PTSD studies, fear and anxiety were not uniquely associated with physiological arousal. Such arousal appears to be associated with negative affect more generally. In some studies, negative emotions were specifically elevated in PTSD participants, whereas in others the negative emotions were associated with the physiological arousal, but not necessarily the PTSD.

In a laboratory project without psychophysiological assessment, Taft, Street, Marshall, Dowdall, and Riggs (2007) used a priming procedure to examine anger in 60 Veterans Affairs Vietnam combat veterans. After listening to (counterbalanced) neutral prime of music or combat sounds, the veterans with PTSD scored higher on state anger than those without PTSD over the experimental priming condition. Those with PTSD also reported greater increases in anger, but not anxiety, following the trauma prime, and did not differ from the non-PTSD group on anxiety.

## Comorbidity Studies and the Location of Posttraumatic Stress Disorder

Psychiatric disorders co-occur in patterns that constitute classes, or spectra, of psychopathology. Recent factor analytic studies of the structure of comorbidity suggest that the covariation of the most common mental disorders can be accounted for primarily by two broad dimensions termed *externalizing* and *internalizing*. Externalizing is a latent dimension of psychopathology that explains the covariation observed in adults between substance-related and antisocial personality disorders (e.g., Krueger, 1999; Krueger, Caspi, Moffitt, & Silva, 1998; Krueger, McGue, & Iacono, 2001) and in children between the co-occurrence of conduct disorder, oppositional defiant disorder, and attention deficit hyperactivity disorder (Coolidge, Thede, & Young, 2000; Dick, Viken, Kaprio, Pulkkinen, & Rose, 2005). Internalizing is the latent factor that underlies the co-occurrence of the anxiety and unipolar mood disorders (Krueger, 1999), which are also known as the “emotional disorders” (Watson, 2005). In several studies, internalizing has

been subdivided into correlated factors termed “anxious-misery” (defined by major depression, dysthymia, generalized anxiety disorder) and “fear” (comprised of panic and phobic disorders; Cox, Clara, & Enns, 2002; Krueger, 1999; Slade & Watson, 2006; Vollebergh et al., 2001). These factors are thought to constitute essential components of the structure of mental illness and have been proposed to form part of a meta-structure for *DSM-V* and International Classification of Diseases (ICD-11; World Health Organization) according to the common factors underlying classes of related disorders.

Recent studies that have examined the location of PTSD within this taxonomy have raised questions about the classification of PTSD as an anxiety disorder and have implications for where the diagnosis should be located in *DSM-V*. For example, Cox et al. (2002) performed a principal components analysis of diagnostic prevalence data from the National Comorbidity Survey (Kessler et al., 1994) and found that PTSD loaded rather weakly on the anxious-misery factor ( $r = .39$ ; composed also of dysthymia, generalized anxiety disorder, and major depressive episode), but did not load significantly on either fear or externalizing in a 3-factor (fear, anxious-misery, externalizing) solution. These results were subsequently replicated in data from an Australian epidemiologic sample ( $N = 10,641$ ; Slade & Watson, 2006) and a U.S. Department of Veterans Affairs study of 1,325 Vietnam combat veterans with a high prevalence of PTSD (Miller, Fogler, Wolf, Kaloupek, & Keane, 2008). Together, results of these studies suggest that PTSD may share more common variance with disorders defined by anhedonic mood and anxious rumination than with those characterized primarily by pathological fear (e.g., panic disorder and phobias) or externalizing.

But does PTSD belong with the anxious-misery disorders in *DSM-V*? Findings from studies on the temporal order of development of PTSD and its comorbidities suggest that it does not. Principles of diagnostic taxonomy suggest that disorders within the same spectrum, thought to arise from a common vulnerability, should be equally likely to precede each other in order of temporal development (e.g., the likelihood of disorder A preceding disorder B should be roughly equivalent to the likelihood of disorder B preceding disorder A). That is, social phobia may develop in an individual with generalized anxiety disorder or an individual with social phobia may later develop generalized anxiety disorder. This does not appear to be true for PTSD where studies of new-onset cases suggest that PTSD exerts a causal influence on most co-occurring disorders, including those of the anxious-misery spectrum with which it is most strongly related. For example, Breslau, Davis, Peterson, and Schultz (2000) examined risk for first-onset major depression during a 5-year assessment interval using data from the Epidemiologic Study of Young Adults ( $N = 1007$ ). Results showed that compared to those who did not experience a new trauma during the 5-year interval, individuals with PTSD due to a trauma occurring during that period were at a greatly increased risk of first-onset major depression

(Hazard Ratio [HR] = 11.71). In contrast, those who were exposed to trauma during the same interval but did not develop PTSD showed no significant increase in risk for major depression (HR = 1.44). Similarly, Brown, Campbell, Lehman, Grisham, and Mancill (2001) examined the temporal sequence of the development of comorbid conditions across a wide range of *DSM-IV* Axis I disorders in 1,127 outpatients and found that in individuals with PTSD, most comorbidity was temporally linked to the onset of the PTSD. Specifically, analyses showed that when PTSD co-occurred with another disorder, the comorbid disorder developed either after or concurrent with the PTSD in 72% of cases with major depressive disorder, 69% of cases with GAD, 68% of cases with alcohol abuse/dependence, 72% of cases with panic disorder/agoraphobia, and 62% of cases with other substance abuse/dependence. In sum, new-onset psychopathology that develops in the wake of trauma rarely precedes or develops in the absence of PTSD. This implies a causal influence of PTSD on comorbid psychopathology and suggests a distinct phenomenology which should be reflected in its diagnostic class membership within *DSM*.

Another principle of psychopathology that should have bearing on the location of PTSD in *DSM-V* is developmental continuity, i.e., the notion that adult psychopathology tends to be foreshadowed by childhood and/or adolescent problems in the same domain. Evidence shows that many adults with anxiety disorders report histories of juvenile anxiety disorders, but they do not typically report juvenile externalizing disorders. The exception to this is found among samples of individuals with PTSD where adult patients frequently have histories of childhood externalizing disorders. For example, Gregory, Caspi, Moffitt, Koenen, Eley, and Poulton (2007) examined data from a large prospective longitudinal study ( $N = 1,037$ ) spanning the ages of 11 to 32 and used follow-back analyses to determine the history of juvenile disorders (i.e., occurring between ages 11–15) in 32-year-old adults with anxiety disorders. Results showed that while adults with most types of anxiety disorders other than PTSD had histories of juvenile internalizing disorders only, approximately 50% of cases with PTSD also had histories of juvenile conduct disorder or oppositional defiant disorder.

Results of twin studies are consistent with these findings and suggest that PTSD shares genetic influences with both internalizing and externalizing spectrum diagnoses, including juvenile conduct disorder (Koenen, Fu, et al., 2005) and substance dependence (Koenen, Hitsman, et al., 2005). Recently, investigators modeled the genetic and environmental architecture of latent internalizing and externalizing dimensions of comorbidity and the relationship of PTSD to each dimension using diagnostic data from 3,372 male–male twin pairs who served in the military during the Vietnam Era (Wolf et al., 2009). Results showed that while PTSD covaried more strongly with disorders of the internalizing spectrum, it also evidenced a significant relationship with the externalizing latent factor defined also by antisocial personality disorder, drug abuse/dependence, and alcohol/abuse dependence.

These findings, and those reviewed previously, suggest that PTSD may arise as a function of latent liabilities towards either internalizing or externalizing psychopathology. This proposition is consistent also with recent studies of personality-based subtypes of PTSD, which have shown that many adults with PTSD exhibit a predominantly externalizing pattern of comorbidity characterized by problems in the domain of impulse–control, antisociality, and substance abuse (Miller, Greif, & Smith, 2003; Miller, Kaloupek, Dillon, & Keane, 2004; Miller & Resick, 2007).

## CONCLUSIONS: ANXIETY OR TRAUMATIC STRESS DISORDER?

The location of PTSD among the anxiety disorders has been a source of controversy since the initial appearance of the diagnosis in *DSM-III*. Our review suggests that fear and anxiety are neither the exclusive nor predominant emotions associated with the development and maintenance of PTSD. Results of comorbidity studies raise further concern about conceptualizing PTSD simply as the manifestation of a vulnerability to anxiety-related psychopathology. We conceptualize PTSD as the product of an environmental pathogen (i.e., a serious adverse life event) operating on individual diatheses that span the spectrum of human variation in vulnerability to psychopathology. This diathesis–stress interaction results in extensive population heterogeneity in the clinical expression of posttraumatic psychopathology, pathological anxiety being just one manifestation of this interaction. To better reflect this, we propose that PTSD be located in *DSM-V* among a class of disorders defined by the causal conditional nature of their relationship to serious adverse life events, i.e., *a spectrum of traumatic stress disorders*. This new class would include the existing diagnoses of posttraumatic stress disorder, acute stress disorder, and adjustment disorder. Consideration should also be given to including “complex PTSD,” a complicated or traumatic grief disorder, and clinically significant trauma-related externalizing reactions not currently captured by any existing diagnostic category. Diagnoses in this class should differ qualitatively from one another and from disorders described elsewhere in *DSM-V* with decisions regarding the inclusion of a given diagnosis based on evidence for its discriminant validity, clinical utility, and clear relationship to a precipitating life event. We believe that this new class of disorders might better capture the heterogeneity of psychiatric disturbances that are manifested in response to serious adverse life events. We expect that it might also foster the development of research and clinical conceptualizations that better reflect the complex relationships between the type and severity of life adversity, individual difference characteristics, and subsequent adjustment. Finally, we hope that the creation of this new class will further the development of treatments for individuals with psychiatric impairments arising from exposure to traumatic stress that fall outside the scope of the PTSD construct.

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